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ORIGINAL MEMOIRS.

AN ANALYTICAL AND STATISTICAL REVIEW OF ONE THOUSAND CASES OF HEAD INJURY.

(Continued from Page 477)

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CONTUSION.—Intracranial contusion may be either of the brain or of the meninges, and in either structure may be limited or diffuse; and when diffuse either one is always accompanied in some greater or lesser degree by an implication of the other.

General Contusion of the Brain.—This condition is usually most marked in the cerebrum, and its manifestations are largely dependent upon injury of that region. If infrequent as an absolutely isolated lesion, it is often practically uncomplicated, being associated only with some minor extension to the meninges. Neither its constancy nor its essential importance have been generally appreciated. This has been due in part to careless and insufficient post-mortem examination, and this again to a still too general acceptance of a theoretical basis of functional disturbance as an adequate explanation of symptoms. The obvious structural alterations are a distention of the parenchymatous vessels, a formation of minute thrombi, the presence of punctate extravasations, and a more or less distinct œdema. All or a certain number of these pathologic

conditions may be seen upon even casual examination. They have been very generally unsought or disregarded, and in the absence of hemorrhage or laceration a contused brain has been pronounced normal.

A more or less intense hyperæmia is often the only gross lesion discoverable. More often careful examination will disclose minute thrombi scattered through the brain surfaces, perhaps generally and uniformly; or they may be in greater part confined to one or more cerebral areas; or, however distributed, but few in number. These thrombi are characteristic of the traumatic form of cerebral hyperæmia. The punctate extravasations are much less frequent in the general than in the limited form of contusions. CEdema occurs in variable amount, sometimes appreciable only by close inspection, or following section and after some delay; or again it may be so profuse that the fluid may be squeezed from the brain as from a sponge; it may be confined to the brain substance or may distend the lateral ventricle; and, as in one instance, it may enormously dilate the *iter e tertio ad quartum ventriculum* and extend into the fourth ventricle. The hyperæmia and cEdema both may be very well marked, but there seems to be no definite relation in their extent; either one may be greatly in excess of the other. The cEdema is more readily explicable as a simple exudation from distended or obliterated vessels; but experiments made by Cannon⁹ seem to establish its dependence upon osmotic pressure.

Since the observation of the present series of cases was begun much has been done to elucidate the intimate pathology of this form of intracranial injury. The primary symptoms are those of shock, though differing in essential particulars from general shock dependent upon irritation of the spinal sympathetic system. The nature of the impression transmitted from the exterior which precedes vascular congestion could formerly be inferred only from symptoms and analogy. The discovery by Obersteiner (1897) that in addition to muscular fibres intracranial vessels even in their minute subdivisions possess filaments which endow them with the power

⁹ Am. Journal of Physiology, 1901-2, pp. 103 *et seq.*

of contraction and dilatation, and that they easily become paretic, confirms an inference that the effects of external violence upon the cerebral sympathetic system may produce a cerebral vasomotor depression similar to that which has been noted for the general circulation through the spinal ganglionic centres. Cannon, however, attributes the primary cerebral anæmia to an increased intracranial pressure which is produced at the moment of injury and is sufficient to check the flow of blood into the brain. The normal vascular condition may be restored without further appreciable aberration, and the patient may recover with only transitory symptoms; but if violence be greater, and the primary nervous impression correspondingly severe, anæmia is followed by hyperæmia. This secondary stage of circulatory disturbance may be recognized at the very earliest practicable post-mortem inspection, and probably begins at once. It may be assumed to result from a paralytic dilatation of the capillaries; and stasis, thrombosis, and punctate extravasation ensue with some progressive increase of intracranial tension, which is still further and more markedly increased by an oedema of the cerebral tissue. The oedema has been very generally ascribed to simple transudation from circulatory arrest, analogous to that caused by obstruction of a vein in the general circulation. The experiments of Cannon show that this result of circulatory defect is less direct. His conclusions are: That theories which regard cerebral oedema as a passive effusion are untenable; that the vascular obstruction deprives the brain of a normal blood supply and thus causes impaired nutrition, especially through a deficiency of oxygen; that the defective nutrition is characterized by active chemical processes which result in increased osmotic pressure and the passage of water into the tissues; that this is the essential cause of the secondary pressure symptoms.

The earlier theories of cerebral changes following external injuries began with the supposition that a hemorrhage, which was regarded as a foreign body, diminished intracranial capacity and thus compressed the brain. Successive areas were supposed to be invaded until the whole brain became involved, and death ensued from obliteration of the bulbar

capillaries and bulbar anæmia. The fact that the same general vascular and nutritive changes occur without the intervention of a hemorrhage or other indications of localized injury is an evidence that they were diffuse from the beginning, and not the final result of a series of extensions through the formation of vicious circles; nor is death always due to bulbar anæmia, nor are all the secondary symptoms those of pressure. It would seem that Cannon might have gone further in deduction. The same deficient blood supply which so disturbed the nutritive processes of the cerebral cells as to cause an abnormal watery transudation could hardly fail to prevent or destroy their normal functional activity. Scagliosi, quoted by Cannon, found, after repeated slight concussions experimentally made, changes in the brain cells varying according to the duration of life. These consisted in varicosities of the dendrites, swelling of the cell body with the formation of vacuoles, and a more and more homogeneous appearance, ending in an almost entire disappearance of the nucleus. If normal structure is essential to normal function such conditions as these are sufficient to account for all attendant symptoms. Primary or secondary loss or impairment of consciousness may be explicable by pressure alone; but aside from so-called pressure symptoms there are others, such as high temperatures, mental decadence, and aphasia, which occur and are indefinitely prolonged, even to the end of life, without indications of pressure.

It may be that defective blood supply alone causes the retrograde metamorphosis of the cells; but it is not justifiable to assume that anæmia is the sole cause, or even that it is invariably operative. It is not improbable that the same primary impression made upon the vasomotor filaments which occasion circulatory derangements may at the same time originate the decadent nutritive processes which go on to cellular disintegration. This supposition is rendered the more probable from the fact that pressure symptoms are only secondary and may be long delayed.

A case in this series heretofore published¹⁰ is suggestive

¹⁰ *Lib. cit.*, Case CXXI.

of cellular changes independent of the circulatory influence. There were well-marked indications of central injury, as loss of consciousness, irregular respiration, unsymmetrical radial pulsations, papillary disturbances, etc.; and death one and one-half hours after admission to the hospital, followed by a rise of temperature of 10.5° post mortem. There was only moderate pial and cerebral hyperæmia, with no punctate extravasations or minute thrombi, but with excessive œdema of all parts of the brain.

In cases of recovery the circulation is readjusted, thromboses, punctate extravasations, and œdema are reabsorbed, and cellular nutrition is restored to normal conditions. There is no means of determining at what pathological point these reparative processes cease to be possible; and the length of time within which they may be deferred and still be practicable is exceedingly uncertain. The severity of symptoms does not always presage their duration; primary or secondary unconsciousness may last for many days, and high temperatures be long continued; but there is a limit of temperature beyond which recovery seems to be impossible and when death is not far away.

Limited Contusion of the Brain.—This does not differ pathologically from the diffuse lesion except, as its name indicates, in the extent of tissue involved. It may be single or multiple, cortical, subcortical, or both, and may vary in size from the fraction of an inch upward, and affect any region of the brain. The hemorrhages are in punctate form and are its most characteristic feature. The œdema, which probably exists, is so soon generally diffused as to be evident only when the area involved is comparatively large.

The lesion when cortical is usually small and situated more often than elsewhere in the temporal region or in the middle or anterior basal fossa. It is obviously a simple bruise, without arachnoid laceration. Its surface is depressed, softened, and variously discolored from a dark red to a yellowish gray. If subcortical the lesion will be larger, marked by more or less scattered punctate extravasations, and more rarely with than without a yellowish stain of the intervening tissue;

when of considerable extent its glistening surface will indicate the existence of œdema. If more than a single lobe is implicated the contusion may be considered general. The superficial injury is almost always an incident of a general contusion and of itself is probably without importance. The subcortical contusion as its extent increases approximates the general lesion. The minute structural changes are the same, and its general symptoms should differ only in degree. Its only special symptoms are those which indicate the inclusion of special centres of control.

LACERATION OF THE BRAIN.—This is the final expression of force in its greatest intensity. It may be single or multiple, cortical or subcortical, primary or secondary, trivial or important. If less absolutely constant than general contusion, it is even more frequently an emphasized and consequently recognized lesion. In its primary form it is most frequently cortical, and like contusion and probably for the same reasons, it most frequently occurs in the temporal region or in the middle or anterior basal fossa; but it is limited to no region of the base or vertex. Subcortical laceration is no less varied in its site. There is no ganglion or convolution which may not be wounded. This series of cases affords examples of laceration, not only of all cerebral and cerebellar regions, but of the pons, medulla, optic thalamus, corpora striata, and olfactory bulbs; and even instances of limitation to the fornix or gyrus fornicatus. Lacerations when superficial, like limited contusions and basal fractures, if not produced at the point of impact are usually situated at the opposite pole of the diameter through which force is applied, and when subcortical are usually in the course of that diameter.

Cortical lacerations may be insignificant in size, or may involve the whole basal aspect of a frontal or temporal lobe. They may occasion but little hemorrhage even when of great extent, or again with a smaller wound the extravasation may cover the whole brain and fill all the basal fossæ—depending upon the size of the vessels ruptured, and it may be upon other conditions. The wound itself is ragged with shreds and

granular detritus of brain matter underlying broken coagula, and after a time may become pultaceous, yellowish or greenish gray in color, and of septic appearance; or in old cases it may sometimes be found lined with a smooth dark-colored membrane which seems to be in process of organization. Its margin is often softened and dotted with miliary extravasations. The arachnoid membrane will be ruptured unless the wound is small. The subcortical laceration is an irregular cavity filled with coagulated or semicoagulated blood, and when cleared of clot is seen to be fringed with long shreds of tissue. It is sometimes of enormous size, excavating an entire lobe, or opposite lobes, or even an entire hemisphere. It may have been originally a small direct laceration, but more probably a slight intracerebral hemorrhage which by its persistence has gradually disintegrated surrounding tissue. It sometimes happens that after the lapse of one or two or more days the disintegrating process involves a vessel of considerable size and a small laceration is suddenly converted into one of great extent, with an equally sudden change of symptoms. These excessive intracerebral hemorrhages may remain enclosed in a mere cortical shell, or they may break through the cortex and spread over the surface of the brain.

A very unusual condition of subcortical laceration was observed in one case of this series. The temporal lobe was largely excavated and the blood had broken through the cortex at only one point. The cavity of laceration was filled in part with a dense black clot, and in part by a detached mass of reddish-brown disintegrated brain tissue, about two inches long, three-fourths of an inch wide, and one-half inch thick. The cortical hemorrhage was extensive and the brain moderately hyperæmic and œdematous. The patient had fallen fifteen feet to the pavement and died the following day.

It is evident from many cases of compound fracture of the vertex, and from others with extrusion of brain matter from the ear, that recovery may follow cortical laceration. It is also certain from the evidence afforded by occasional necropsies that immediate recovery has followed these lacera-

tions. There were four such instances noted in the present series of cases, in each of which death had resulted from more recent injury. These were:

I. Deep laceration of inferior surface of a temporal lobe, one inch in its diameters, with irregular outlines, lined with a greenish-yellow viscid substance, and surrounded by an area of yellow softening. Previous history unknown.

II. Small cavity in parietal lobe communicating with lateral ventricle, the surface of which was softened, watery, and of a greenish color, and was found later to consist of a fine neuroglia without cells and with only a few capillaries. The intracranial injury had been received four years previously.

III. Shrunken depressed cystic cavity at upper extremity of fissure of Sylvius, extending into cerebral substance to within 5 mm. of the roof of the lateral ventricle; 3.1 cm. in depth and 1.4 cm. in breadth; surrounding tissue sclerosed, and convolutions contracted and atrophic; general sclerosis with marked frontal atrophy. The patient was said to have been injured ten years previously.

IV. Canal lined with blood-stained membrane extended from the cerebral surface between frontal and ascending parietal convolutions to the site of a bullet projecting through the cortex near the median line. The patient was shot four years previously. There was also a laceration of the first and second temporal convolutions, with clean surface and rounded edges, at the site of a trephination made some months before death.

The reparative changes which must precede possible cicatrization are: Formation and organization of a cyst wall, liquefaction and absorption of the clot, contraction of the cyst, and its final obliteration. The formation of a cyst and the liquefaction of the clot have been often seen in operations for epilepsy following former intracranial injuries, and the subsequent contraction of the cyst is noted in one of the instances just cited; but traces of its final obliteration have not been observed in the course of necropsies made in the present series of cases.

It is not probable that large subcortical lacerations often end in the recovery of the patient, since these cavities are so rarely seen in later post-mortem examination. It may be assumed that they do not cicatrize, since there is no record of a cicatrix having been found post mortem with the history of

a former intracranial lesion—a very small cicatrix might escape detection, but hardly one of appreciable size. How often smaller lacerations, whether cortical or subcortical, have occurred in recovering cases which are subsequently lost to view it is impossible to estimate. The lack of distinctive symptoms which permit the differentiation of general contusion from laceration, as well as the fact that laceration is always attended by general contusion, prevents positive knowledge.

CONTUSION OF THE MENINGES.—Meningeal contusion may be either of the *dura mater* or *pia mater*, or as in case of other lesions they may complicate each other.

Contusion of the *dura mater* is usually manifested by hemorrhage derived from rupture of the dural vessels or venous sinuses, or by sinus thrombosis. In exceptional cases the *dura* may be congested or even inflamed. The dural hemorrhage is usually the only source of an epidural hemorrhage which occurs independently of cranial fracture. Thromboses were occasionally found in each of all the sinuses. These were sometimes of very recent, or possibly even post-mortem formation; and sometimes, as shown by their firmness and decoloration, antedating death by a considerable period. The larger number of them are to be ascribed to direct injury rather than to contusion. This is evident in cases in which the sinus wall is torn by an osseous fragment or contused in a line of fracture. The superior longitudinal sinus is thrombosed in this way not very infrequently in connection with fracture of the vertex, and the lateral or cavernous sinus more rarely with fracture of the cranial base. Instances of thrombosis from direct injury of the sinus wall are to be found in cases of this series heretofore published, and might be cited from among those of later occurrence.

Thromboses which occur independently of direct injury, pressure, inflammatory process, or infection, have been considered difficult if not impossible of explanation. A recognition of the far-reaching effects of intracranial contusion renders their occurrence entirely comprehensible. The minute thrombi which it occasions in the minute vessels of the brain

and pia mater are simply replaced by a large thrombus in a correspondingly larger vessel. The denser structure of the sinus wall prevents dilatation as it does rupture or punctate extravasation; and the paucity of nutritive processes affords no sufficient grounds for œdema. The adequacy of this explanation is made still more manifest by exclusion. There are not only cases in which no injury of the sinus wall is discoverable, but there are others in which there is no fracture in the same region of the cranium, or even no fracture at all—and this in the absence of inflammation or infection, and with a clot of obviously ante-mortem formation. The number of cases of this character is not large, probably not more than a half dozen in the whole series. The notes of necropsies which follow are illustrative.

I. No fracture; no hemorrhage; no lacerations; general cerebral contusion indicated by hyperæmia, moderate œdema, and thrombosis of minute vessels which involved the basal ganglia, pons, and cerebellum; no punctate extravasation; decolorized thrombus in each lateral sinus extending into inferior petrosal sinus and jugular vein; thrombosis of ophthalmic vein.

II. Direct basal fracture confined to middle fossæ; epidural hemorrhage in one middle fossa; cortical hemorrhage covering opposite temporal lobe and superior cerebellar surface; general pial and cerebral hyperæmia and thrombosis; firm thrombus in each lateral sinus.

III. Direct basal fracture confined to one side of the cranium; dura mater greatly congested in its posterior two-thirds; small white clots in anterior half, and red clots in posterior half, of the superior longitudinal sinus; thick subarachnoid purulent effusion at base, and seropurulent effusion at vertex of the brain; pus in middle ear on side of fracture.

There was no reason to suspect in any case save two the existence of a special sinus lesion prior to its disclosure in the course of operation or post-mortem examination. In these two instances there was paralysis of all the ocular muscles, and in one case impaired sensation in the course of distribution of the first and second branches of the fifth nerve. Diagnosis of a thrombus in the cavernous sinus, compressing the third, fourth, and fifth nerves in their passage through its walls, was made by Dr. P. A. Callan. There was fracture of the cranial base, and other suggestive conditions, in both cases; but abso-

lute confirmation of the diagnosis was lacking in the absence of opportunity for post-mortem inspection.

It might perhaps be expected that thrombosis of the lateral sinus would be recognized from symptoms, as it sometimes is in cases of middle-ear disease; such has not been the case, whether from urgency of other conditions or for other reasons.

Contusion of the Pia Mater.—This is the more distinctive form of meningeal contusion, and more closely resembles the cerebral injury of which in some measure it always forms a part, and to which it is usually proportionate in degree, but not necessarily in extent. It is often general and uniform when at the same time the cerebral lesion is essentially confined to one lobe or one hemisphere. Like cerebral contusion it is limited or diffuse, and is indicated by hyperæmia, thrombosis, punctate extravasations, and œdema; unlike the cerebral lesion it may occasion a diffuse hemorrhage without gross laceration of tissue. This hemorrhage as was previously stated occurs in patches, in a thin sheet spread over the vertex, or in quantity sufficient to break through upon the cerebral surface or into the arachnoid cavity. It is the form in which pial contusion most frequently becomes a source of distinctive danger; and it was in noticeable amount in about 30 per cent. of all the necropsies made. The punctate extravasations are more numerous than in cerebral contusion, often become petechial, and largely replace the minute thromboses. The greater proneness to hemorrhage in pial contusion is explicable by the structural peculiarities of the pial membrane. Its vessels are larger and more numerous, and the loose areolar meshes in which they subdivide afford inefficient support in time of stress from active dilatation.

The pial œdema, which is the analogue of the cerebral transudation, may be confined in the areolar interstices of the membrane as it is held in the connective tissues in case of œdema elsewhere, and diffused over a considerable part of the vertex or of a hemisphere; or it may be circumscribed in scattered areas of very limited extent. It more characteristically occurs as a subarachnoid effusion which may cover the

whole brain, but more frequently the vertex alone; or a hemisphere or even only one or two lobes; and it is a clear or slightly opalescent fluid. It is not only of much less frequent occurrence than pial hemorrhage, but is probably still less frequent as an essential or even largely contributive factor in determining a fatal result. It does not seem to modify in any recognizable degree symptoms of severe cerebral contusion, with which it is always complicated. Any operation based upon its supposed existence and importance therefore must be purely empirical.

There is no reason to suppose that the origin of these structural changes is other than the same shock of external violence impressed upon the cerebral vasomotor centres which produced similar changes in the brain substance.

SECONDARY PATHOLOGICAL CHANGES.—The vast majority of cases of intracranial injury end in recovery or death from the direct effect of the primary lesion; but in the minority, certain other pathological conditions may intervene between the primary contusion and the ultimate issue of the case. These are inflammatory or degenerative, and as they affect the brain substance are: *softening*, *sclerosis*, and *abscess*.

Softening.—This does not occur in large areas involving the central regions of the brain, as happens in idiopathic cases resulting from atheromatous degeneration, or from embolism or thrombosis of large vessels. It is always practically confined to the cortex, and is often but not always inflammatory. It is then only an incident in a meningeal inflammation with which it may be co-extensive, and is a nutritive defect explicable through a community of vessels by direct anastomosis. The hemorrhagic form, which often occurs in multiple areas, is also a part of an injury of wider extent, and was noted in many cases of pial contusion with petechial hemorrhages which involved the cortex, or with superficial lacerations or limited contusions. It did not usually extend for more than 1 to 3 cm. into the brain substance, though in one instance it was prolonged through a parietal lobe from the fissure of Sylvius to the posterior commissure. Its superficial extent was small,

or at the most but very moderate—from a few millimetres to in one instance the whole of the inferior portions of the three frontal convolutions, which were infiltrated with blood.

The softening of the margin of large subcortical lacerations which occurred when death was not immediate, was variously inflammatory, hemorrhagic, or œdematous.

A fourth form of superficial softening was occasionally observed, which resulted from contact with subarachnoid or ventricular effusions; and in one case the surface of the third left convolution was softened and eroded by the pressure of a hæmatoma. These secondary changes may be discovered at the earliest period of post-mortem inspection.

Sclerosis as a sequel of cerebral contusion does not differ anatomically from the same inflammatory condition when idiopathic or congenital. It is the result of a subacute grade of inflammation with progressive increase of neuroglia, concurrent cellular destruction, and ultimate atrophy. It is attended by other meningeal and cortical lesions which perhaps measure the severity of the deeper contusion. It may begin soon after the receipt of injury, but probably only in exceptional cases, though it should be said that it was not especially looked for in cases which terminated in a very early period. The beginning of sclerotic change was noted in one instance in a general increase of neuroglia when death had occurred on the seventh day; and in another case, when death was on the twenty-fifth day, the left frontal lobe had already become perceptibly atrophied. How frequently it may exist in those cases in which with decadent symptoms the patient survives intracranial injury for a considerable length of time is quite uncertain, since post-mortem examination is not usually made. It may effect the brain generally or be limited to a single hemisphere, or to a single lobe as it was in some of the cases cited in illustration of frontal localization.¹¹

The sclerosed part in accordance with the extent of structural change will only be slightly firmer than normal, or indurated and atrophied in some greater or lesser degree.

¹¹ Phelps: Am. Journal of Medical Sciences, April-May, 1902; March, 1906.

A case of sclerosis and atrophy of the temporal lobe previously cited¹² was especially notable. The primary symptoms were delirium and a single convulsion; six months later an operation for fractured patella was followed by wild delirium and convulsions; and one month later still, a second operation was again followed by delirium and convulsions, and by death on the second day. The right temporal lobe was found to be indurated, pigmented, shrunken to less than one-third of its normal size, and of dense fibrous structure with no trace of resemblance to brain tissue. A cortical laceration, $3\frac{1}{2} \times 1\frac{1}{2}$ inches in its diameters, involved the whole of the second and third and a little of the first temporal convolutions. There was also a laceration of the anterior border of the right frontal and one of the second and third orbital convolutions of the left frontal lobe. All of these lacerations were necrotic.

In another case also previously cited¹³ the patient survived more than two years. In this instance atrophy was limited to the anterior part of the brain, and to a very great extent to the left frontal lobe; and there was direct evidence of primary lesion of the brain tissue.

Secondary inflammations have essentially the same pathological history whether the brain tissue or its membranous investment is the part affected.

The direct infection of compound fractures, and through them of subjacent intracranial structures by continuity and a community of vessels, is part of the common history of all infected wounds. In this way limited or diffuse septic inflammations of the dura mater, arachnoid membrane, and cerebral cortex or even subcortex, may successively occur. Their mode of development and their subsequent extension do not differ from those of the same pathic condition when otherwise produced.

The indirect infection of distant parts which has been considered in some detail in a former study of the subject¹⁴

¹² *Lib. cit.*, Case CXLVII.

¹³ *Lib. cit.*

¹⁴ *Lib. cit.*, edition of 1900, pp. 69 *et seq.*

causes inflammations of the same character as those which originate by direct transmission through contiguous structures. These are: A parenchymatous inflammation of the subcortical brain substance resulting in abscess, usually limited, but in at least one instance diffuse, and a limited or diffuse inflammation of the arachnoid membrane, in which the inflammatory products are found in the arachnoid cavity or in the subarachnoid spaces and in the tissues of the pia mater. Their dependence upon an invasion of pathogenic germs from without or from some other part within the body is beyond question in cases of cerebral abscess and of the suppurative form of arachnitis. It has been averred that traumatic cerebral abscess is invariably attended by cranial or extracranial wound. This contention is disproved as an absolute rule by at least exceptional cases. There are other channels of invasion, notably the vascular, which are no less accessible to these germs after an intracranial injury than when the intracranial contents are still intact.

There is in all cases in which suppurative inflammation is of this indirect character a direct traumatic lesion which is essential, and a later infection at its site which is accidental. These are the fundamental factors in their pathology—a diminished power of resistance in a part weakened by a primary traumatic lesion, and its natural selection as a point of attack by predatory pathogenic germs. Observation and inference have so well established this relation of the primary to the secondary lesion that it may be assumed to exist in every instance until proof to the contrary be adduced.

Notwithstanding all the possibilities of infection—the existence of a superficial wound or fracture, the frequent surgical want of cleanliness, the neglect or imperfection of early or late aseptic or antiseptic treatment—its actual occurrence is infrequent.

Cerebral abscess occurred in twelve cases, one of which was superficial and an incident in the history of a neglected compound fracture; and another which was more properly a diffuse encephalitis than an abscess. They constituted practically 1 per cent. of the total number of 1000 cases.

SUMMARY OF ABSCESS CASES.

	Cases	Recovered
Abscess complicating fractured base.....	4	1
Abscess complicating fractured vertex.....	8	3
Recoveries		33%

In at least 3 of these cases and probably in two others abscess had already formed.

Probable Route of Infection.—Through compound fracture, 9; through ear, 2; through sloughing points in the scalp, 1. Associated arachnitis, 6 (probably primary, 4).

Region Infected.—Frontal lobes, 5; frontal lobes and lateral ventricles, 3; lateral ventricles, 1; parietal lobe, 1; occipital lobe, 1; diffuse, 1.

The case last mentioned is so unusual in character, if not unique, that it will be cited at length.

CASE XXIX.—A lad ten years of age was knocked down and run over by a truck. On admission to Bellevue Hospital he was unconscious; temperature 96°, pulse 122, respiration 28. His face was contused and bleeding and his left femur fractured. There was hemorrhage from the nose, mouth, and right ear; and hæmatemesis. On the third day he had double internal strabismus, frontal headache, and great restlessness. On the fourth day in the afternoon his temperature rose from 99.8° to 103.6°, and afterwards remained high with morning remissions and afternoon exacerbations. No note was made of his mental condition until the sixth day, when he is said to have been somnolent with a cessation of headache and lessened restlessness and strabismus. On the ninth day there was some cervical rigidity; on the eleventh day lumbar puncture was made without result; and on the thirty-first day he was sent home.

During his stay at Bellevue he was at times quiet, rational, and somnolent; but oftener noisy, restless, irritable or delirious, and crying throughout the day or night because of pain in his head; vesical and rectal control was retained; vomiting or inability to retain nourishment was only an occasional occurrence. No medication was employed except an occasional cathartic and on two occasions $\frac{1}{4}$ gr. codeine.

He was in care of a private physician for six days, and is said

to have emaciated and to have been "apparently in a state of coma."

On the thirty-eighth day he was admitted to St. Vincent's Hospital. His temperature then was 102° , pulse 112, and respiration 30. He was very restless, hyperæsthetic, and could be aroused only with difficulty. His mental condition remained unchanged until the seventh day, and from that time he was conscious, but manifested no sign of intelligence; vesical and rectal control was lacking at all times; the muscular condition was normal except for one day, when the extremities were rigid on attempt at flexion of the larger joints. His temperature became normal on the third day, rose to 102° on the fifth, fell to normal on the sixth, and was afterwards subnormal till his death on the ninth day—the forty-seventh from the time of injury.

Ocular examination by Dr. Callan disclosed neuritis in the right eye; the retina of the left eye was concealed by an old corneal opacity. Lumbar puncture yielded a turbid fluid which contained no pus.

On the fourth day, while his earlier history was yet unknown, he was trephined on the supposition that there might be a subacute arachnitis with large serous effusion. The dura mater was found to be closely adherent to the bone, apparently from pressure, but there was no effusion into the arachnoid cavity. The cortical surface was tense and feebly pulsating, and its vessels were distended.

LESIONS.—*Fracture*.—Essentially confined to the cranial base; closed fissure began in the left superior occipital fossa, ran inward through left inferior occipital fossa, crossed the median line and inner third of right petrous portion, became open, and ran through the right middle fossa to terminate in the centre of the squamous portion. *Intracranial hemorrhages*, none. *Arachnoid cavity* contained no fluid. *Limited contusion*—a single one which was small, superficial, and of the inferior surface of the left temporal lobe. *Brain substance* upon superficial section was markedly hyperæmic and œdematous; and upon deeper section the whole right parietal and occipital lobes were found to be converted into a single enormous cavity filled with seropus; the left occipital lobe was similarly excavated and was filled with turbid serum and a thick green pus, the latter in large proportion; the frontal lobes were hyperæmic and œdematous, having escaped infection.

Ventricles.—The lateral ventricles were enormously distended with a thin seropurulent effusion which held in suspension flakes of thicker pus. This same fluid filled the fourth ventricles, excavated a wide canal through the pons varolii, largely accumulated in the posterior basic fossæ, and extended into the spinal canal. The posterior border of the cerebellum and its median fissure were superficially infiltrated with pus. The infecting germ was the *Staphylococcus pyogenes aureus*. There can be little doubt that the primary lesion was a general contusion and œdema of the brain substance, and that it was this watery effusion which was the direct seat of infection. The general character of the infiltration and predominance of serum over pus, and the œdema of regions not infected, point to this conclusion.

Pachymeningitis is the most infrequent form of traumatic intracranial inflammation. There were four cases altogether—two which were external and two which were internal. The two cases of pachymeningitis externa were in each diffuse, and resulted from infection through an external wound engrafted upon an old idiopathic inflammation of alcoholic origin. The instances of pachymeningitis interna were part of a general septic inflammation of the intracranial contents; and the infection was doubtless through the pia mater and the arachnoid membrane.

The puffy tumor described by Percival Pott, in which a cranial contusion was followed by necrosis and a limited pachymeningitis with abscess, has no longer reason to exist and has probably ceased to occur.

Acute arachnitis occurred in twenty-one cases, nearly twice as many as there were with cerebral abscess, or about 2 per cent. of the total number of 1000.

Complicated with basal fracture.....	14
Complicated with fracture of vault.....	3
Complicated with simple intracranial injury.....	4

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There were extracranial wounds in thirteen cases, including two of the four in which there was no fracture, and four in which the wound was gunshot.

The route and manner of infection seems evident in a majority of cases, as shown in the following table.

ACUTE ARACHNITIS WITH FRACTURE OF THE CRANIAL BASE.	
Infection before admission (positively known).....	1
Mastoiditis	1
Penetrating fracture of orbit.....	2
Complicated fracture involving ethmoid cells.....	1
Complicated fracture—wound, stuffed with soiled rags.....	1
Complicated fracture—gunshot (ball extracted from nose in one)	2
Complicated fracture with hemorrhage from the nose.....	2
A fracture through anterior fossæ (with hemorrhage from the nose)	1
Wounds of the scalp.....	1
Inexplicable	2
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<i>With Fracture of the Vault.</i>	
Complicated fractures (1 homicidal—multiple; 1 gunshot)	3
<i>With Simple Intracranial Injuries.</i>	
Mastoiditis	1
Unknown injury one month before admission.....	1
Recent infection before admission.....	1
Wounds of scalp.....	1
<hr/>	
	4

Six cases, which comprise compound fractures, simple wounds of the scalp, and fractures with hemorrhage from the ear, afford an appreciable route for infection; but with no appreciable reason for its occurrence. They were, so far as known, given the same aseptic care as the much greater number of others which escaped; whether infected before admission or by some lapse in treatment afterward must remain in doubt.

The two cases which are enumerated as inexplicable were without wound or extracranial hemorrhage. One was admitted to the hospital on the fourth day after receipt of injury, unconscious and with a temperature of 104° ; and the other, without obtainable history, had spastic muscles and a slightly subnormal temperature. They died respectively on the second and third days after admission. The first upon necropsy was found to have

cranial fracture extending from a point beneath a temporo-occipital hæmatoma through both middle fossæ and the sella turcica, excessive cerebral hyperæmia and moderate œdema, and a seropurulent effusion in an area not more than one inch in diameter. The second had fissures confined to the posterior basal fossæ, laceration of the inferior surface of a frontal lobe with marked hemorrhagic softening, cerebral hyperæmia; subarachnoid serous effusion over the entire base of the brain, and a small area of seropurulent effusion over each frontal lobe. In each case the effusion was insignificant in amount and the mild infection seems from the history to have occurred prior to the patient's coming to the hospital.

The arachnoid inflammation was general, covering the whole vertex and base, in only five cases; in twelve others it covered one-half or more of the brain, anteriorly, laterally, or at the base or vertex alone; and in the three remaining cases it was confined to one or more very small areas. The effusion in some cases was wholly purulent, and in others was diluted with serum and occasionally mingled with shreds or granules of fibrin.

The essential factors in the causation of acute arachnitis, as of cerebral abscess, are a primary contusion and a secondary infection. There is this single pathological difference, that while the abscess is usually circumscribed the arachnoid supuration is often diffuse, and may involve the entire membrane. Its strict limitation in other cases to small areas of contusion demonstrates the effect of a primary lesion in determining the site of infection as in the following instances:

I. Seropurulent effusion confined to an area of not more than one inch in diameter upon the posterior border of the cerebellum.

II. Seropurulent effusion confined to two areas, each two by two and one-half inches in diameter and situated over a frontal lobe.

III. Four areas each about two inches in diameter with arachnoid opacity and subarachnoid sanguinolent effusion.

IV. Several small purulent effusions in different cerebral regions.

V. Posterior half of the pia mater and arachnoid mem-

brane covering the vertex excessively hyperæmic; anterior half opaque, with subarachnoid seropurulent effusion flattening the convolutions and obliterating the sulci.

In each of these instances the primary contusion was evident beneath the subarachnoid effusion.

Subacute Arachnitis.—This occurred in thirteen cases. There was also a small number of cases in which a serous effusion might have been to some extent inflammatory, but in which its origin was still questionable. The inflammation was general in all but one, and in that one extended over two-thirds of the cerebral surface. There was no fracture in nine cases, though in eight there was extracranial wound with or without fracture.

The anatomical appearances were very like those of meningeal contusion with non-inflammatory subarachnoid effusion. The distinction was made by the cloudiness or turbidity of the serous effusion and by the opacity of the arachnoid membrane. These characteristics were more or less well defined in different cases, or either one might be absent. In one instance the serous effusion was wanting while there was great arachnoid opacity over the whole superior cerebral and cerebellar surfaces; in another the serous effusion was turbid with no loss of arachnoid transparency; and in a third there were patches of fibrin over the left frontal lobe and along the median fissure, and a membranous exudation covering the pons, medulla, and inferior cerebellar surface, with a turbid serous effusion confined to the lateral ventricles. Sometimes the membranes were locally adherent to each other, and sometimes when inflammation was long continued in connection with a cerebral sclerosis they were much thickened.

Histological examination was made in only a minority of cases, and in them there was no evidence of pus—only small round cells which also infiltrated the cerebral cortex. This grade of inflammation, which is amicrobic, is ascribed by Gouley¹⁵ to the irritation of dead atoms which have failed of absorption. This explanation is based upon Bland-Sutton's theory of inflammation.

(To be continued.)

¹⁵ See *lib cit.*, edition of 1900, p. 85.